disease (Eysenck, 1990). Our own work (Grossarth-Maticek, Eysenck & Vetter, 1988) certainly supports this view.

About the relevance of stress to CHD, there exists a good deal of confirmatory evidence (e.g. Hanson, 1987; Kaplan et al., 1978.) Brindley & Rolland (1989) have recently reviewed the evidence, concluding that "an increased control of metabolism by the 'stress' or counter-regulatory hormones....is a common feature of (CHD) risk factors. Particular emphasis was placed upon the action of the glucocorticoids - (which) can decrease energy expenditure and, together with insulin, promote energy deposition. These observations provide a partial explanation for the metabolic changes that can accompany the risk factors and clarify why they interact in promoting atherosclerosis." (p.459.)

As regards the cancer-prone personality, this has often been described as appeasing, unassertive, overco-operative, overpatient, harmony seeking and conflict avoiding, compliant and defensive (Baltrusch et al., 1988). The two most frequently noted characteristics are (1) suppression of emotional expression, and denial of strong emotional reaction; and (2) failure to cope successfully with stress, and the reaction of giving up, linked with feelings of hopelessness and helplessness (Eysenck, 1985; Baltrusch, Stengel & Waltz, 1988). This type has sometimes been called "Type C", to distinguish it from the coronary heart disease-prone Type A, and the healthy Type B. (Temoshok, 1987.)

Many studies have given support to the notion of a "cancer-prone" type, but most of these have been studies comparing individuals suffering from cancer with controls of one kind or another (Eysenck, 1985). Three major prospective studies were mounted to overcome the possibility that the cancer might have caused differences in

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personality, rather than that personality and stress were risk factors for cancer. These three studies have been described in detail by Grossarth-Maticek, Eysenck & Vetter (1988), and we will only quote the major results. The first of these studies was carried out in a small country town in Yugoslavia, where every second household was studied, and the oldest inhabitant used as proband. (There was also an additional small sample of highly stressed people, and occasionally when the oldest inhabitant was a female, Yugoslav macho attitudes made it necessary to take into the sample the oldest male. These deviations from the research plan had the effect of worsening the outcome, rather than improving it, but the figures given below are for the total sample.)

The other two studies were carried out in Heidelberg, and used in the first instance a normal, fairly random sample, with certain age and sex limitations imposed. The second Heidelberg sample was a stressed sample; its members were nominated by members of the normal Heidelberg sample as being highly stressed; they came largely from friends and relatives of members of the first sample.

Members of these samples were interviewed and given a personality/stress questionnaire; they also gave information concerning their smoking and drinking habits; and blood pressure and blood cholesterol were investigated, in order to have some medical information relevant to these possible risk factors.

The personality/stress interviews and questionnaires were used to assign each proband to one of four types. Type 1 was believed to be cancer-prone, showing the characteristics outlined above. Type 2 was believed to be coronary heart disease-prone, again showing a set of characteristics quite different from those of the cancer-prone type. Type 1, in brief, is characterized by under-stimulation; persons of

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this type show a permanent tendency to regard an emotionally highly valued object as the most important condition for their wellbeing and happiness. The stress produced by the continued withdrawal or absence of this object is experienced as an emotionally traumatic event. Type 1 individuals fail to distance themselves from the object and remain dependent upon it. Thus individuals of this type do not achieve success in reaching the object, and remain distant and isolated from this highly valued and emotionally important object. Great stress is produced by the failure to achieve nearness to the highly valued person, success in a highly valued occupation, or whatever.

Persons of Type 2 (CHD-prone) rather show <u>over-arousal</u>; they show a continued tendency to regard an emotionally highly important object as the most important cause for their particular distress and unhappiness. Rejection by the object, (if a person), or failure to reach it (as in the case of occupational success) is experienced as an emotional trauma, but persons of this type fail to achieve disengagement from the object; rather they feel more and more helplessly dependent on the object. Thus persons of this type remain in constant contact with these negatively valued and emotionally disturbing people and situations, and fail to distance themselves and free themselves from dependence on the disturbing object, reacting with anger (open or suppressed) and aggression.

There is a third type, Type 3, characterized by ambivalence. This type shows a tendency to shift from the typical reaction of Type 1 to the typical reaction of Type 2, and back again. Members show a permanent tendency to regard a highly valued object alternately as the most important condition for his own wellbeing, and is the main cause for their own unhappiness. In individuals of this type, we have an

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alternation of feelings of hopelessness/helplessness and of anger/aggression. Possibly because of this alternation of quite different reaction types, this particular type of proband seems to be relatively safe from both cancer and coronary heart disease.

Persons of Type 4 are characterized by personal autonomy, and in some ways resemble Type B of the Friedman-Rosenman typology, or to the "hardy" personality of Allred & Smith (1989). For persons of this type there is a strong tendency to regard their own autonomy, and the autonomy of the persons with whom they wish to be in contact, as the most important condition for their own wellbeing and happiness. This enables them to experience realistically the approach or avoidance behaviour of the object of their desires, and thus enables them to accept the autonomy of the object. An inventory to determine which type a proband belongs to has been reproduced in the paper by Grossarth-Maticek, Eysenck & Vetter (1988).

Table 4 shows deaths, and causes of deaths, from various diseases by type of personality in the Yugoslav sample. The crucial figures are the percentage of deaths from cancer in Type 1 (46.2%), and the percentage of deaths from coronary heart disease in Type 2 (29.2%). The figures for both are obviously much lower for Types 3 and 4, and the data give substance to the view that personality and stress can be used to predict deaths from cancer and coronary heart disease. It will also be clear that Type 3 has a high proportion of deaths from other causes, whereas Type 4 has much the largest percentage of probands still alive.

Table 4 here.

Table 5 shows deaths from various diseases by types of

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personality in the Heidelberg normal sample. Here the number of deaths is very much smaller, because the sample is younger than was true of the Yugoslav sample (the average age of the Yugoslav sample was 62 years, that of the Heidelberg normal and Heidelberg stressed sample was 50 years of age.) Clearly, cancer and coronary heart disease are diseases of old age, and the ten year difference in age makes a considerable difference to the number of deaths expected. Nevertheless, the figures clearly show again a relationship between cancer and Type 1, and coronary heart disease and Type 2.

Table 5 here.

Table 6 shows deaths from various diseases by type of personality in the Heidelberg stressed sample. Here the number of deaths is much closer to that in the Yugoslav study, in spite of the difference of ten years in age; as the two Heidelberg samples are very similar in all other respects, it seems reasonable to attribute this larger numbers of deaths to the stress experienced by this group. In the Heidelberg normal sample, 88.6% were still alive at the end of this ten year follow-up; in the Heidelberg stressed sample, only 54.3% were still alive. This difference is one of the best pieces of evidence for the importance of stress in causing death that exists in the literature.

Table 6 here

The data can be inspected more directly in the form of Figures 3, 4 and 5, which show clearly how close is the relation between personality type and disease.

Figs. 3, 4 and 5 here

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We must now consider the importance of smoking for the type-cancer relationship. Table 7 shows a number of cancer deaths, other deaths and total deaths for non-smokers and smokers of Type 1, as compared with individuals of the other three types. Among non-smokers, as expected, there are very few deaths from lung cancer, but of the 13 that occur, 10 occur in persons of Type 1. For smokers, there are 74 deaths, only 6 of which occur in persons other than Type 1. These results give rise to an association between Type 1 and lung cancer of $P = 0.0001$ for both the samples considered when a correction has been made for differences in smoking habits according to the Mantel-Haenszel (1959) formula. It is clear that quite independent of smoking, individuals of Type 1 are cancer-prone, as compared with individuals of Type 2, 3 and 4. (See also Grossarth-Maticek, Eysenck & Vetter, 1988.)[1]

Table 7 here.

This table also makes clear that there is a synergistic interaction between smoking and typology. The only group which has a high proportion of deaths from lung cancer is that of smokers of Type 1; smokers not of Type 1, and non-smokers either of Type 1 or of the other types have negligible rates of cancer deaths. Of the two factors, smoking and personality, personality seems to be the stronger. Of 735 smokers not of Type 1, 6 only were found to have died of lung cancer; this figure is not very different from 3 non-smokers not of Type 1 who died out of the 850 individuals who were non-smokers. Clearly smoking appears to represent a danger to health as far as lung cancer is concerned only for individuals of Type 1.

The data may deserve a slightly more formal analysis, following

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Footnote, p. 78.

1. There are several explicit or implicit replications of these studies, showing similar relationships. A review of earlier studies has been given by Eysenck (1985). More recent studies are those of Kune et al. (in press), Quander-Blaznik (in press), Beek (no date), and Wirsching et al. (1981). Of particular interest is the work of Baalen & de Vries (1987), who showed that in patients with extensive metastases who recovered without operations or chemotherapy, all had personalities resembling those of Type 4.

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the traditional methods of epidemiology (Perkins, 1989). The background factor for the two populations (Yugoslav and Heidelberg stressed) taken together is .35% for lung cancer mortality (N=850). For personality in non-smokers it is 2.89%, giving an excess of 2.54% (2.89% - .35%), which may be called the personality effect. For smoking in personality other than Type 1 the effect is .45% (.80% - .35%), i.e. about a fifth of that of personality. The combined effect of smoking and personality is 15.21% (15.56% - .35), which is 5 times the effect expected from simple addition of the smoking and personality effects (.45% + 2.54% = 2.99%). Thus synergism produces a 500% increase in cancer mortality from smoking and personality.

This calculation is taken over 2,374 people, giving 77 cases of lung cancer mortality; the numbers are clearly not large enough to take the resulting calculations as anything but a very rough and ready guideline. Also it may be objected that the calculation brings together two rather unlike populations (Yugoslav and German), differing in age and stress. However, both groups give similar results when analysed independently, and hence may not be too dissimilar for the purpose of analysis. (The Heidelberg normal group had too few cases of lung cancer to be included.)

We have carried out similar analyses for other risk factors (blood pressure, blood cholesterol), and compared these with smoking as a risk factor. Details and figures are given elsewhere (Grossarth-Maticek, Eysenck & Vetter, 1988). These analyses were carried out for all three samples, i.e. the Yugoslav, the Heidelberg normal and the Heidelberg stressed sample. The results are quite similar, no matter which dependent variable, which organic variable and which place of investigation is considered. Table 8 show the results.

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Table 8 here.

(1) The organic variable has different relevance for mortality depending on the psychosocial type. Its relevance is greatest with that type which itself has the greatest specific mortality, i.e. Type 1 for cancer, and Type 2 for coronary heart disease. In other words, the psychosocial types are relevant, not only for mortality, but in a similar way for sensitivity to organic risk factors.

(2) The psychosocial types do show differences with respect to the organic variables, but these differences cannot explain away the relevance of the types for mortality; according to the figures for mortality, type-specific mortality differences when adjusted for differences of the organic variables are still highly significant.

These data suggest several conclusions. Psychosocial variables, and particularly personality type and stress, are important in mediating deaths from cancer and coronary heart disease. These personality variables are more influencial than physical factors like smoking, blood pressure, and cholesterol, in the ratio of roughly 6-1.

Finally, personality and physical factors interact in a synergistic fashion. These are important findings for any attempt to prevent deaths from cancer and coronary heart disease.

The findings that risk factors in cancer and coronary heart disease act synergistically, not by simple addition, is so important that these findings may be amplified by two studies not yet published. One of these is summarized in Table 9.

In this table, we are dealing with the results of a study in which we compare death rates of probands having 1, and 2, and 3, or all 4 of four different risk factors. The risk factors were (1) smoking (more than 20 cigarettes per diem, for over 10 years), (2)

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heredity (at least one first-degree relative suffering from or died of lung cancer), (3) chronic bronchitis, and (4) stress, i.e. probands of Type 1 or 2. Not all combinations of risk factors could be found in sufficient numbers, but the data show very clearly the synergistic effects of multiplying risks.

Table 9 here.

It will be seen that in these probands, who were on average between 51 and 54 years old at the beginning of the study, 13 years later none had died of lung cancer of those who only showed one risk factor. Of those showing 2 risk factors, only about 1% died of lung cancer. Combinations of 3 risk factors showed quite elevated death rates for lung cancer, varying from 7.6 through 9.8 to 20%. Combinations of four risk factors raised the death reate from lung cancer to 31%, demonstrating the strong synergistic effect of multiplying risk factors.

Of particular interest in this chapter is the group of 4 risk factor probands put in brackets; they had received prophylactic behaviour therapy (BT), and accordingly had a death rate from lung cancer only about 1/3rd as high as the group of 4 risk factor probands who received no therapy. Thus even for those most exposed to lung cancer prophylactic treatment is possible, and can be efficacious. A later chapter will deal with the prophylactic effects of behaviour therapy.

In another study, using 684 selected probands having various combinations of 0 to 7 risk factors (smoking, stress, genetics, bronchitis, use of depressive drugs, contact with automobile emissions, contact with asbestos). The results are shown in Fig. 6,

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for various combinations of these factors; again the synergistic or multiplicative nature of these risk factors in producing lung cancer will be clear.

Fig. 6 here.

These studies add to the earlier evidence (Grossarth-Maticek, 1980, 1989; Grossarth-Maticek, Eysenck & Vetter, 1988) and are indicative of synergistic action. This notion of multiple risk factors interacting in a synergistic fashion is crucial for a proper appraisal of methods of prevention for cancer and coronary heart disease. The failure of monocausal intervention efforts (McCormick and Skrabanek, 1988; Eysenck, 1986) is almost certainly due to concentration on single risk factors, like smoking, rather than a more wide ranging consideration of many different risk factors. As we have seen, psychosocial factors are of particular importance, and may present a more useful point of departure for prophylactic action. Support for this hypothesis is given in the next section.

It should not be assumed that the notion of synergistic action finds support only in the Grossarth-Maticek & Eysenck studies. In a recent paper Perkins (1989) has argued that: "Interactions among the major coronary heart disease risk factors of smoking, hypertension, and elevated cholesterol may contribute substantially to the p prediction of CHD risks over and above the sum of the independent risks due to their factors." (p.3.) He surveys results which show that "the interaction of smoking and cholesterol and of hypertension and cholesterol may each as much as double the risks of CHD which might be expected if these factors acted only additively." (p.3.) He also comments on "the strong possibility of an interaction between chronic psychosocial stress and elevated cholesterol." (p.3.) Other

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authors who have argued in favour of synergistic interaction effects are Kleinbaum et al. (1982), Rothman (1974), Rothman & Keller (1972), Kannel et al. (1986) and Saracci (1987); Perkins (1987) and Walker (1981) as well as Kooperman (1981) and Perkins (1985) have also added to the methodological analysis of synergistic effects.

An example will illustrate methods and results. Kannel et al. (1986) have reported data on over 300,000 white males selected for inclusion in the MRFIT study already mentioned. For men between 35 and 45 years of age, in the lowest quintile of diastolic blood pressure (<76 mm Hg), the six year risk of CHD mortality among non-smokers without elevated cholesterol (i.e. background risks) was 0.6/1,000, while the risks due to smoking alone was 1.0/1,000 and the risk for non-smokers with cholesterol in the highest quintile (above 2.44 mg/dl) was 2.0/1,000. If the effects of smoking and elevated cholesterol were only additive, the risk of those with both factors present would be 2.44/1,000, i.e. the background risk of 0.6, plus the risk attributable to smoking (0.4) and to cholesterol (1.4). The actual mortality rate for this subgroup, however, was 4.8/1,000, i.e. twice that expected on an additive model, indicating an excess risk of 100%!

This is clearly an important area of research, and the concepts involved may explain why single risk factors so often fail to give consistent positive results. It is certainly completely inadmissible to use simple univariate analyses for risk factor studies in epidemiology; multivariate analyses are an absolute must (Wilhelmsen, Wedel & Tibblin, 1973). Certainly smoking emerges as a risk factor in conjunction with other risk factors, rather than by itself. For this, as well as for other reasons already adumbrated, talk of smoking as "causing" cancer or CHD is scientifically meaningless.

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The established facts of synergistic interaction between risk factors also have profound consequences for prophylaxis and prevention generally (Perkins, 1989). The relative failure of quitting smoking to reduce cancer and CHD mortality may be due to the fact that such quitting would only have any pronounced effects in people with more than one risk factor present. For people with no other risk factors except smoking, quitting smoking would have no effect on mortality, and their inclusion in any study would disguise any impact that quitting smoking might have on mortality.

This certainly is the implication of the published data summarized above, and particularly of our Table 7. Quitting smoking in persons of Type 4 would only have a minimal effect, if any, on mortality.

For readers who would like to have some more detailed information on the Grossarth-Maticek personality types, an Appendix has been provided which lists the questions in a short version of the questionnaire which has been used in more recent studies (Grossarth-Maticek, Eysenck, 1990). In these later studies, 2 more types have been added to the 4 types already mentioned, Type 5 relating specifically to rheumatoid arthritis, as well as to cancer, and Type 6 to drug addiction. Type 5 is characterized by rational-antiemotional behaviour, and Type 6 by anti-social, psychopathic behaviour.

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(7) Intervention studies in cancer and coronary heart disease.

In the preceding section we have attempted to demonstrate that psychosocial factors like stress and personality are important risk factors in the causation of cancer and coronary heart disease, and that specific personality constellations, reacting to stress, are particularly cancer-prone and coronary heart disease-prone. These relationships, although much stronger than those linking smoking to cancer and coronary heart disease, are still only correlational, and it is well-known that correlations cannot be used directly to infer causation. The best way of indicating that the relations observed are indeed causal is by some intervention method which brings to bear an experimental paradigm on the problem in question. We have attempted to do this by using methods of behaviour therapy (Eysenck & Martin, 1987) in an attempt to alter the behaviour of the cancer-prone or the coronary heart disease-prone person in the direction of our healthy Type 4. In other words, we have attempted to increase autonomous behaviour, and reduce the proband's dependence on other people, or his/her acceptance of situations which lead to negative consequences. In this section we will give a brief discussion of the methods used, and then go on to an evaluation of the effects of using these methods in a prophylactic manner. (For a more general discussion of prophylactic methods, see Aeberhardt, 1989.)

The therapy developed by us contains many features which are familiar, such as Wolpe's method of desensitization, Lazarus's development of coping mechanisms, social skills training, and others. The method has been called 'Autonomy Training', or 'Creative Novation Behaviour Therapy'. The major aim of the treatment is to stimulate

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an individual to look towards the long-term positive results of different types of behaviour and self-evaluation (Grossarth-Maticek & Eysenck, in press). Thus it is the aim of the treatment to increase behaviours which lead to long-term positive consequences, even though this may involve some short-term negative consequences. Conversely, the patient should learn to avoid behaviours which lead to long-term negative consequences, even where these may be associated with short-term positive consequences.

Having briefly described the methods of prophylactic behaviour therapy, we must now consider their application to a variety of probands of Types 1 and 2, i.e. people prone to cancer and coronary heart disease respectively. Three major studies have been done by us, using respectively long-term individual therapy, group therapy, and bibliotherapy conjoined with short individual therapy. These studies will be discussed with respect to death, cause of death (as shown on the death certificate) and incidence, i.e. the diagnosis of cancer or coronary heart disease made by the patient's physician, and ascertained with the prior permission of the patient. All the studies were carried out in Heidelberg (West Germany), and the therapist in each case was Professor Grossarth-Maticek who originated the methods used.

Probands in the first of these studies, using long-term individual therapy, were 100 individuals, half male, half female, who were categorized as Type 1 (cancer-prone) on the basis of interview and questionnaire data. Their mean age was around 50 years. The second group of 92 individuals, similarly selected, but all of Type 2, were chosen to study the effects of behaviour therapy on coronary heart disease. The therapy consisted of roughly 30 hours of individual therapy, as outlined at the beginning of this chapter.

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The results are shown in Table 10. It will be seen that both for cancer and for coronary heart disease, the number of deaths is very significantly less for the therapy group than for the control group. The same is true for incidence which is roughly half in the therapy group as compared with the control group. These figures suggest very strongly that behaviour therapy can prevent death from cancer or coronary heart disease, or at least postpone it for a very considerable period of time. It is of course possible that the surviving members in the therapy group will ultimately die of cancer or coronary heart disease, or at least some of them will; only a much longer follow-up, until all the participants of the study are dead, can answer this question. What we do see is that 13 years after the initiation of the study, many more members of the therapy group are alive than of the control group.

Table 10 here.

It should also be noted that details of all probands were transmitted to two independent observers prior to ascertaining death and cause of death, or incidence, in each case. In addition, a random sample of interviewers was interviewed by an independent observer to check on the collection of interview and questionnaire data. These precautions were taken as a routine measure to certify the objectivity of the data collection.

Our second study is concerned with group behaviour therapy. In this case, groups of between 20 and 25 probands were formed, and administered group behaviour therapy for periods of between six and twenty-five hours, each group meeting lasting between two and three hours. Duration was determined by the members of the groups themselves, and hence varies to a considerable degree. Matching was

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carried out as before, and members of the group were followed up over an 8-year period. Results are given in Table 11.

Table 11 here.

It will be seen that group therapy, very much like long-term individual therapy, benefitted the therapy group, as compared with the control group, by reducing the mortality from cancer and coronary heart disease, and decreasing the incidence. Given the relative unreliability of diagnoses as recorded on death certificates, the most impressive figure perhaps is that of the proportion still living, which indicates the power of behaviour therapy to prevent cancer and coronary heart disease. Again the procedure, including all the death certificates, was checked by an independent observer.

Our third study was concerned with a special kind of bibliotherapy, which centred on a text entitled: "How to Achieve Emotional Independence and a Healthy Personality". This text was introduced to participants in an hour long introduction by Professor Grossarth-Maticek, who also outlined the application of the principles contained therein to the individual proband. After a week or so to give the proband time to read and understand the text, and try to apply it to his/her own condition, specially trained students went to discuss this application of the message contained in the text to the individual's circumstances, for three one-hour periods. What we are dealing with here, then, is not just bibliotherapy, but in addition some four hours of individual therapy.

In this study the therapy group was constituted of 600 probands, matched as before with a control group of 600. Again probands were allocated on a chance basis to therapy or control. Within the control group, a small group of 100 was exposed to a placebo

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treatment, i.e. using a psychoanalytic text outlining Freudian ideas concerning the origins and prevention of cancer. Table 12 shows the results of the study. It will be seen that the placebo group does not differ significantly from the control group, but the therapy group is superior to the control group both with respect to smaller number of deaths from cancer and coronary heart disease, and lower incidence of these two diseases. The data show that even a relatively short-term application of behaviour therapy, together with bibliotherapy, can have a marked effect on mortality and incidence of cancer and coronary heart disease.

Table 12 here.

More detailed information concerning these studies is given by Eysenck (1988a, b; 1989; Grossarth-Maticek, Bastiaans, and Kanazir, 1985; Grossarth-Maticek, Eysenck & Vetter, 1988; Grossarth-Maticek, Schmidt, Vetter & Arndt, 1984). These communications also contain information on the possibilities of prolonging life even after terminal cancer has been diagnosed. It seems that the use of behaviour therapy can delay deaths from cancer and almost double the duration of survival.

It is sometimes said that the results of behaviour therapy in cancer and CHD, either prophylactically or in prolonging life, are too good to be true, yet there is much outside evidence for the efficacy of different types of psychological therapy on cancer and CHD, some of which reports results even better than those described above. To take only the most recent study, Spiegel et al. (1989) was able to double the life expectancy of female patients with metastatic breast cancer, a result even better than similar studies reported by us (Eysenck,

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1988a, b). In Spiegel et al's control group, life expectancy was 18.9 months, while in the therapy group it was 36.8 months. Such findings should be seen in the context of studies looking at the influence which psychosocial factors and interventions have on the immune system, which presumably mediates the effects of life-events, stress and therapeutic psychological intervention, and the occurrence and rigour of cancer. Before turning to a consideration of this evidence, it may be useful to consider studies sugesting the possibility of prophylactic intervention in coronary heart disease-prone probands.

Johnston (1989) has reviewed the literature in a very critical spirit, but concludes that the evidence for the view that stress management may reduce coronary heart disease through the lowering of a large number of stress related risk factors by moderate, or even small, amounts "is patchy, but a much stronger case can be made than would have seemed possible only ten years ago". (p. 277.) The cautious optimism of this quotation is mainly based on the contributions of Friedman (1987), Friedman et al., (1984, 1986), Gillet et al. (1985), Lovibond et al. (1986), and Patel et al. (1985); most convincing are the data presented by the recurrent coronary prevention project. If the results are less striking than those reported by Grossarth-Maticek, Eysenck & Vetter (1988), this may be due to the fact that the validity of the Type A concept is probably much lower than that of the Grossarth-Maticek Type 2, i.e. the coronary heart disease-prone type (Booth-Kewley & Friedman, 1987). It is also noteworthy that the theory linking stress with cancer, and stress-reduction with cancer survival, has been elaborated much more convincingly (through the innervation of the immune system) than can be said of the relation between stress and stress management, and coronary heart disease.

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Bennett & Carroll (1990) similarly conclude a review of the evidence by saying that stress management techniques "not only reduce individual risk factors, they can also reduce mortality and morbidity to CHD." (p.1.) They also conclude that "risk factors combine multiplicatively, and small decreases on a number of risk factors may reduce the risk of CHD more than if only one risk factor is targeted (Johnson, 1989; Perkins, 1989), as in most medical interventions." (P.81.) These conclusions are very much in line with our own studies.

Finally, we may quote the conclusions drawn by Taylor (1990) in his review of Health Psychology: "Research that examines whether or not psychological and social factors are involved in health and illness has largely made its point..... Investigations have addressed the direct impact of stress and other psychological states on physiological processes, the impact of psychological and social factors on risky health practices, and the impact of psychological and social factors on how people respond to potential illness states, such as whether or not they engage in appropriate illness behaviour. As such, the field has advanced to an unprecedented level of complexity in research investigations... Health psychology affords the opportunity to look beyond particular disorders to the broad principles of thought and behaviour that cut across specializations of diseases or problems studied to elucidate more fundamental psychosocial mechanisms." (p.46.)

Some of the resulting complexities are illustrated in the work of Brown & McGill (1989) and Scheier et al. (1989). The importance of optimism in the recovery of patients from coronary artery disease by-pass surgery was highlighted by Scheier et al., demonstrating again the interaction of psychological states, personality and medical

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illness of recovery. Brown & McGill demonstrated the complex interaction of personality traits like optimism, which is closely linked with extraversion (Eysenck & Eysenck, 1985) and stress.

Positive life events are generally believed to have beneficial effects on health, but apparently this is only true when probands have a positive self-concept (optimism). Brown & McGill (1989) outline an identity disruption model of stress, which holds that an accumulation of life events that are inconsistent with the self-concept leads to illness. Thus positive life events in probands with a negative, pessimistic self-concept were predicted (and found) to predict the development of illness over time. Clearly, over-simplified concepts of "stress" may lead to erroneous predictions, and may account for many failures to replicate in the literature.

The findings of Brown & McGill find support in an experimental study by Brebner (1990) who showed that introverts tend to generalize experiences of failure, extraverts experiences of success, thus developing pessimistic or optimistic personality traits. Brebner considers such generalization of failure experiences as an important form of stress, but of course it is not usually mentioned in traditional stress inventories. It is noteworthy that the characteristics of cancer-prone probands agree to a considerable extent with those of introverts, while CHD probands tend to show more the characteristics of extraverts (Eysenck, 1990). These apparent relationships between predisposition to disease and well-established personality types are well worth following up along theoretical and experimental lines.

A brief outline may here be given of the way the connection between personality/stress and disease may be mediated by hormonal and physiological factors. A more detailed outline is given elsewhere

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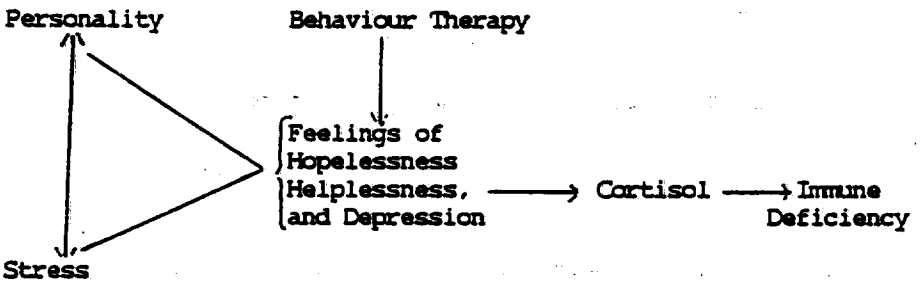
(Eysenck, 1986). The figure below illustrates the assumed causal pathway. Personality (Type 1) and stress combine and interact to produce feelings of helplessness, hopelessness and depression; these in turn produce hormonal and other reactions of which cortisol is here given as the representative (others are the endogenous opiates, ACTH, etc.). These in turn produce immune deficiency, which allows budding cancers to develop. The well-established fact that immune reactions can be conditioned along classical lines suggests one possible way such reactions may be learned. (Ader & Cohen, 1975; Solvason, Ghanta & Hiramoto), 1988.) There is a good deal of evidence to support such a model.



The model owes much to a similar one by Solomon (1987, in press; Solomon & Moos, 1964; Solomon, Levine & Kraft, 1968) who has argued powerfully for the concept of an "immunosuppression-prone" personality (Solomon, 1985). Having surveyed the literature, he produced first 35, and later another 30 postulates, many of which are relevant to our discussion. The main postulates of interest here are given below, numbered in sequence.

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Solomon's Postulates, 1987.

1. Enduring coping-style and personality factors (trait characteristics) should influence the susceptibility of an individual's immune system to alteration by exogenous events, including reactions to events. (Thus, an "immunosuppression prone" behavioral pattern is hypothesized.)

2. Emotional upset and distress (state characteristics) should alter the incidence, severity, and/or course of diseases that are immunologically resisted (infectious and neoplastic) or are associated with aberrant immunologic function (allergic and autoimmune).

3. Severe emotional disturbance and mental dysfunction should be accompanied by immunologic abnormalities.

4. Experimental behavioral manipulation (for example, stress, conditioning) should have immunologic consequences.

5. Experimental manipulation of appropriate parts of the central nervous system should have immunologic consequences.

6. Hormones and other substances regulated or elaborated by the central nervous system should influence immune mechanisms.

7. Biochemical and functional similarities might be expected between the substances modulating the function and reactivity of the

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central nervous system (neuropeptides) and the substances with comparable effects on the immune system (cytokines).

8. Behavioral interventions (such as psychotherapy, relaxation techniques, imagery, biofeedback, and hypnosis) should be able to enhance or optimize immune function.

9. Altered CNS neurotransmitter receptor site sensitivities felt to be associated with mental illnesses should be reflected in lymphocyte receptors.

10. The "functional" modes of expression of CNS and immune system should be similar.

Linn, Linn & Jensen (1981) have shown that stress and anxiety are associated with depressed immunological response. Levy et al. (1985, 1987) found that natural killer (NK) cell activity in breast cancer patients was strongly correlated with psychosocial stress indicators which accounted for 51% of the baseline NK activity variance. Warburton & La Via (1987) found that tendency towards avoiding stress was the best predictor of immunocompetence. Green and Green (1987) reported that relaxation increases salivary immunoglobin A^1. Bandura et al. (1988) found that perceived self-inefficacy in exercising control over cognitive stressors activated endogenous opioid systems. Kiecolt-Glaser et al. (1984) found that distressed and lonely probands had significantly higher cortisol levels and a lower level of natural killer cell activity. Glaser et al. (1986) discovered stress-related impairments in cellular immunity, and Glaser & Kiecolt-Glaser (1985)

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found that even "relatively mild stress" depressed cellular immunity in healthy adults. Kiecolt-Glaser et al. (1984) found that high scorers on stressful life events and loneliness had significantly lower levels of NK cell activity. Herberman (1988), Irwin, Vale & Britton (1987), Nemeroff et al. (1984) and Rou et al. (1988) found impaired immune reaction in depressed groups, and Linn, Linn & Klimas (1988), Arnetz et al. (1987), Glaser, Kiecolt-Glaser, Speicher & Holliday (1985) and Shavit et al. (1989) found impaired immune reactions to stress. Jemmott and Magloive (1988) found that stress lowered salivary concentrations of S-IgA, while social support increased them. Grossarth-Maticek and Eysenck (1989) found that behaviour therapy significantly increased the percentage lymphocyte count in terminally ill women suffering from cancer, and also increased their survival time. Pennebaker et al. (1988) found that self-disclosure improved cellular immune functioning. Kiecolt-Glaser et al. (1985) found an enhancement of immunocompetence by relaxation and social contact.

Irwin, Daniels, Bloom, Smith and Weiner (1987) have shown that life events can cause depression, and reduce the effectiveness of the immune function. Similarly, Murphy, Monson, Sabol and Leighton (1987), in a prospective study of 1003 adults, found a significant correlation between depression and mortality. Rodin (1980, 1986) showed that appropriate psychotherapy reduced depression and cortisol level through psychotherapy. Dabbs and Hopper (in press) showed that cortisol level correlated with anxiety, depression and high heart rate.

Finally, the relationship between mood and the immune system response has been established in a series of studies (e.g. Baker, 1987; Dillon and Baker, 1985; Linn, Linn & Jensen, 1984; McClelland et

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al., 1989, 1985; Morgan, 1989, and Stone et al., 1987). Animal studies, too, have contributed to the formulation of the model (e.g. Glaser et al., 1985; Landenslager et al., 1983; Borysenko and Borysenko, 1982; and for a review, Justice, 1985.)

The studies quoted are only among the most recent; for reviews of the older and perhaps less convincing material the following are suggested: Jemmett and Locke (1984), Miller (1983, 1985), Baker (1987), Kennedy et al. (1988), Testima (1986), Pletnikoff, Faith, Murgo and Good (1986), Korneva, Klimento and Shkhinek (1985), Antoni (1987) and Steptoe (1989). Taking all the published data together, they do seem to support the sort of model suggested by Dilman and Ostronmova (1984) and Eysenck (1986), and briefly outlined above. There is evidence that (1) personality and stress produce immunodestructive substances in the bloodstream; (2) that these substances do have such an immunodestructive function, and that (3) behavioural manipulations can reverse this process. Thus there appears to exist at least a preliminary model to explain along causal lines the effectiveness of behaviour therapy in prophylaxis for cancer, and in prolonging life in cancer sufferers.

There is one apparent objection to this argument. As Zanderman, Costa and McCrae (1989) have shown, there is no evidence in a nationally representative sample for any correlation between depressive symptoms and cancer morbidity. The answer to this is very simple. Depression is a multi-faceted set of symptoms, like fever, which may have diverse causes and relate to different disorders; the difference between reactive and endogenous depression is perhaps the best known. The type of depression referred to in our theory is sub-clinical, and might be defined as "hopelessness depression" (Alloy, Abramson, Metalsky and Hartlage, 1988). This concept is largely based

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on the work of Seligman (1975) and Abramson, Seligman and Teasdale (1978), and is essentially a cognitive diathesis-stress theory of depression (Alloy, Clements and Holden, 1985). According to this theory, "a proximal sufficient cause of depression is an expectation that highly desired outcomes are unlikely to occur, or that highly aversive outcomes are likely to occur, and that no response in one's repertoire will change the likelihood of occurrence of these outcomes." (Alloy et al., 1988 p.7.) It is in this sense that the term has been used in our research. Other varieties of depression may or may not be relevant, and it is important to note that animal work too has emphasized the importance of differentiating between escapable and inescapable shocks, and the vital contribution of predictability (Miller, 1981.)

As far as coronary heart disease is concerned there is less material to review, but sclerosis is an obvious intermediary. Grossarth-Maticek and Eysenck (in press) have reported a study in which 100 cancer-prone and 92 coronary heart disease-prone probands had the degree of sclerosis in the fundus of the eye measured on a 3-point scale by a leading ophthalmologist; before and after therapy (for a randomly selected 50% of probands in each case) and at similar points of time for probands in the control group. Fig. 7 shows the results. Type 2 probands have significantly higher levels of sclerosis than Type 1 probands, and the therapy group a significantly lower degree of sclerosis; more so for CHD-prone Type 2 than for cancer-prone Type 1 probands. This experiment is in urgent need of replication.

Fig. 7 here.

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It is of course necessary to preserve caution in interpreting these results. As in the case of epidemiology generally, there are no singular causes producing single results. Thus Cobb and Rose (1973) showed that air traffic controllers have a higher incidence of hypertension than controls, and produce the condition at a younger age. This could be interpreted as a direct effect of the greater stress under which air traffic controllers work. However, hypertension is strongly related to alcohol consumption in air traffic controllers (De Franks, Jenkinson & Rose, 1987). It is possible that the drinking may be the important determinant, as alcohol intake is known to raise blood pressure (MacMahn, 1987.). Complications of this kind are the rule in this field, rather than the exception.

Clearly the Grossarth-Maticek and Eysenck data concerning the prophylactic effects of behaviour therapy for cancer and coronary heart disease are equally subject to this caution. We have shown (Grossarth-Maticek, Eysenck, Gallasch, Vetter and Frentzel-Beyme, in press) that after therapy there is a very significant shift from Type 1 or Type 2 behaviour to Type 4 behaviour; there is no change in smoking habits. However, as Pearl (1925) has pointed out, there is a style of life which embraces many different behaviour patterns, and changing one may change many others, one of which, or any combination of which, can carry the burden of changes in cancer-proneness or coronary heart disease-proneness. We are only at the beginning of the scientific study of these complex nomological networks, and should not pretend to a greater or more secure understanding of these causal effects than is reasonable under the circumstances.

Eysenck (1989) has discussed the social implications of this type of work. Clearly preventive medicine is much more humane in its consequences, as well as much cheaper, than traditional medicine which

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waits until the disease has manifested itself before attempting any intervention. Many practical, social and ethical problems are raised when we consider the possibility of introducing methods of this kind for the prevention of cancer and coronary heart disease into the social systems at present concerned with health, but it is time that the issues were taken seriously, and debated in a meaningful fashion. These issues are too serious to be left to epidemiologists.

What is most important from the point of view of this book, of course, is the demonstration of the heightened probability of a causal relation between stressed personality, on the one hand, and cancer and coronary heart disease, on the other. The failure to demonstrate such a relationship between smoking and disease, shown in previous sections, is the most vulnerable point in the orthodox view. These results show that it is possible to demonstrate causal relations following accepted scientific methodology, and to establish personality and stress as important risk factors in cancer and coronary heart disease.

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(8) Summary and Conclusions.

In spite of all the criticisms that may justifiably be made of modern epidemiological methods and findings, it is possible to make some fairly definitive statements which characterize the present status of research in the field covered by this book.

(1) It is clear beyond doubt that there is a debate going on concerning risk factors in cancer and coronary heart disease, particularly smoking. On the one hand we have a large group of physicians, epidemiologists and others who claim that smoking is the major cause of cancer and coronary heart disease, and that if only we could stop people smoking large numbers of lives could be saved. On the other hand we have a large number of physicians, epidemiologists, oncologists and other scientists who believe that what we are dealing with is a multiplicity of risk factors, interacting in complex ways, of which smoking is only one, and not by any means the most important. The Press has almost entirely suppressed representation of this alternative view, and has given rise to the completely unjustified and unscientific belief that smoking is the main and almost the only risk factor for cancer and coronary heart disease. It seems about time that the truth regarding the existence of this debate was publicly acknowledged, and that the irresponsible exaggerations concerning the effects of smoking ceased to be published and broadcast.

(2) There is now little doubt that smoking has certain positive effects which account for its popularity, and the curious fact that even people who believe that smoking causes disease still continue to smoke. Among the positive features of smoking which have now been firmly established by research are its ability to raise the cortical

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arousal level, and thus counteract boredom and fatigue; its ability to make anxious and tense persons relax, thus reducing the impact of negative emotions; and its power to increase attention and vigilance, thus enabling people to work longer and harder, and more efficiently, than they could do otherwise. These are important advantages, which should be set against the disadvantages of smoking. There does not seem to be any truth in the allegation that smoking is an addiction, even if that term could be defined in such a way as to have a scientific meaning. People maintain the smoking habit because of the positive effects of smoking on their psychological wellbeing; if we substitute alternative behaviours which serve the same needs, smoking can readily be given up as being no longer required.

(3) The relative importance of smoking as a risk factor, compared with psychosocial and other risk factors, has received a good deal of attention. Roughly speaking it would appear that personality and stress are 6 times as important as smoking in the statistical correlation between disease and risk factors. This does not mean that smoking is necessarily harmless, but it certainly puts into perspective its possible influence on cancer and coronary heart disease.

(4) What emerges with particular clarity from all the work that has been summarized in these pages is the importance of psychosocial factors, i.e. stress and the individual's reaction to stress. Of all the risk factors considered, this is perhaps the most important, showing the strongest correlation with death from cancer and coronary heart disease. It is curious, considering the long history of theories concerning psychosocial factors, going back for over 2,000 years, that the medical profession has on the whole failed to pay much attention to the importance of stress and personality in the causation

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of disease, and tends to reject claims that stress is a real killer. The evidence is too strong now to reject such claims out of hand.

(5) The only model which is remotely adequate to contain the known facts in this field is a multifactorial synergistic model, i.e. one in which many risk factors are included, and where these risk factors act in a synergistic, (i.e. multiplicative), rather than in an additive fashion. Thus in our own work smoking has always been found to be uncorrelated with cancer or with coronary heart disease in groups where there was no stress, or where a healthy personality counteracted the stress and managed to cope with it. Positive correlations with disease do appear in populations of Type 1 or 2, i.e. populations where stress is endemic, and coping behaviour is inadequate.

(6) So far we have discussed only correlations between risk factors like smoking, stress, personality, etc., and disease; it is well known that correlation does not imply causation, and that statistical association may suggest but does not prove causation. The best demonstration that a causal model may be indicated is furnished by intervention studies. If we believe that smoking causes cancer and coronary heart disease, then clearly quitting smoking should reduce the risk of cancer and coronary heart disease. As we have shown, the evidence does not suggest that this is so. Where quitters are self-selected, they already differ profoundly in their state of health from continuing smokers, and any differences in outcome are more likely to be due to this difference than to the effects of quitting. Where quitters are part of a therapy group, their future health does not, or only slightly, differ from that of continuing smokers. The evidence is confused at best, but it certainly does not support the wildly excessive claim that has been

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made for the beneficial effects of quitting smoking.

(7) The position is quite different with respect to psychosocial factors, where the evidence now seems conclusive that the application of the methods of behaviour therapy to people suffering from stress and inadequate coping mechanisms can prevent cancer and coronary heart disease, when groups receiving such help are compared with groups not receiving it. Thus not only are psychosocial factors statistically much more closely related to disease, but there is evidence for a causal influence here which is absent in the case of smoking.

(8) Clearly there must be intermediary factors intervening between stress and personality, on the one hand, and disease, on the other, and the discovery of such factors is one of the major tasks for future research. It seems reasonable to assume that the major intermediary in the case of cancer is the immune system, and there is evidence that behaviour therapy can improve the efficacy of the immune system, while stress has the opposite effect (Eysenck, 1986; Rodin, 1984, 1986). Similarly, there is evidence for a marked influence of stress and behaviour therapy on the degree of sclerosis shown by individual probands (Grossarth-Maticek et al., in press). With this whole field of research being so young, this area has not been sufficiently researched to come to any very definite conclusion, but these are suggestions which are certainly worth following up.

It would not be right to pretend that the position in which we find ourselves is a very comfortable one, or that our knowledge is anything like as adequate to the challenges of disease as it ought to be. One of the major reasons for this unsatisfactory position is that research money has gone into areas which have proved unrewarding. We may with advantage remind ourselves that the Multiple Risk

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(9) Epiloque.

The question of risk factors for cancer and coronary heart disease, and the relative importance of smoking as a risk factor, is clearly an important scientific problem which should be solved by the use of traditional scientific methods. Yet we have found that conclusions have often been based on inadequate methodology, doubtful assumptions, inappropriate statistics, and inadmissible extrapolations. It seems that evangelical fervour, dogmatic certitude and propagandist intentions have only too often led to a premature crystallization of spurious orthodoxies which has set back the factual study of the subject and delayed the finding of proper answers by many years. It is unfortunate that the issue has become clouded by an anti-smoking crusade which pays scant regard to the facts of the case, but uses facts selectively to prove a conclusion already predetermined on a priori grounds.

In this the whole controversy resembles similar controversies which have ranged around issues like the role of cholesterol in CHD, or that of fluoridation of the water supply. In both cases, publicity campaigns were waged in order to get people to substitute margarine for butter, change the whole diet to lower cholesterol levels, increase the fluoride level of the water supply, and quite generally change people's habits in line with supposititious benefits to their health. Had these benefits been real, and had these changes in fact produced better health and longer life, the fanatical support given these measures might be understandable. However, as already noted, there do not seem to be any obvious benefits to be derived from a

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cholesterol-low diet, and in fact it might be linked with increasing risks of cancer. Similarly, fluoride in the water supply is now stated to provide little if any benefit as far as tooth decay is concerned and may indeed have the opposite effect. Furthermore, it has been suggested that it may be linked with increasing risks of cancer.

I cannot pronounce on these developments as far as their medical background is concerned; the new developments may have as little substance as the old. They may, however, have the beneficial effect of making lay readers, and possibly even medical readers, less prone to take seriously any pronouncements of epidemiological "experts" based on insufficient evidence and grossly defective studies. What is being criticized is not the individual scientist who draws the wrong conclusion from his data, or who commits errors of one kind or another in his experimental methodology, or his statistical analysis. To err is human, and all scientists are prone to blunders of one kind or another.

But scientists should be modest in their claims, should refuse to exaggerate the certainty of their conclusions, and should always be open to correction and the consideration of contrary evidence. In particular, they should refrain from advocating far-reaching social actions on the basis of doubtful, fallible, disputed and insufficient evidence. Equally, they should not pretend that contradictory data do not exist, that anyone who doubted the correctness of their arguments was acting on improper motives, or that it was irresponsible to argue against their firm convictions. Yet the propounders of the low cholesterol diet, and the advocates of fluoridation, have been guilty of all these misdemeanours, as have those who have condemned cigarette-smoking out of hand as being responsible for the majority of

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deaths from cancer and CHD.

Here, as with cholesterol and fluoride, scientists have been guilty of suppressio veri and suggestio falsi, of neglecting to inform the public of facts contrary to their beliefs, of public statements going way beyond the established facts, of emotional propaganda not based on solid evidence, and altogether of fanatical publicity-seeking quite alien to the received model of the genuine scientist. There is no culpability in being wrong, but there is culpability in perpetuating error in the face of contradictory evidence, in bigoted and zealous over-enthusiasm, and in prematurely urging major social intervention on the basis of doubtful and contradictory evidence.

The grossly exaggerated claims concerning the degree of risks attending cigarette smoking in the causation of cancer and CHD must stand with the grossly exaggerated claims concerning cholesterol and fluoride as a warning to scientists to avoid jumping on bandwagons, join publicity campaigns, or advocate premature social action. As Claude Bernard warned a hundred years ago: "In ignorance, abstain!"

There is one additional matter that requires discussion in this context. The premature claims for the preventive possibilities of a reduced cholesterol diet in relation to CHD, fluoridation in relation to tooth decay, and giving up smoking in relation to cancer and CHD, and the widespread publicity these have received through the media, have suggested to the layman that a far greater certitude attends to these claims than can be justified scientifically, as we have seen. The recent findings that these claims are in fact unfounded, and that fluoridation, or indulgence in a low cholesterol diet may in fact harm rather than benefit the individual, have led to bewilderment, and a feeling, only too well justified, that "expert" opinion on medical

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matters is quite untrustworthy, and that the constant reversals of recommendations for a healthier living emanating from such sources indicate a lack of true knowledge, and a partisanship unworthy of scientists. As Le Fanu (1987) has pointed out, in the 30s we were advised by nutritionists to eat more eggs and butter, and drink more milk. A few years later all these nutriments became anathema, in the interests of a low cholesterol diet. Now we are being told that this was all a mistake, and that there is no harm in eggs, butter and milk, and that they may even be good for us. Clearly something has gone wrong very badly when violently opposite recommendations are made on the apparent basis of "scientific" evidence, and the public has a right to be bewildered, and to doubt if such advice is really based on fact rather than passing fancy. This undermines public faith in the objectivity of science, and the truth-value of scientists' published beliefs; it is, indeed, the old story of the boy who cried 'Wolf'. Having cried wolf so often, only to retract the statements of the alleged danger, nutritionists, epidemiologists and other medical specialists are now in danger of being disbelieved even when their warnings are only too well justified.

Worse yet, warnings which are not justified by the state of our present knowledge may themselves increase stress, and lead to very serious health consequences. Our study (Grossarth-Maticek & Eysenck, 1989) has shown that such fears are indeed only too real, and that the constantly repeated media threat that smoking kills has in fact become a self-fulfilling prophecy! Thus the lack of responsibility shown in making publicly unjustified statements about the evil consequences of smoking is underlined by the actual consequences of making such statements, namely the deaths of numerous people frightened literally out of their lives by the media and their grossly and very partial

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accounts of the evidence (Clark, 1989). When the assertions made by apparently authoritative bodies and persons, such as the Royal College of Physicians or the Surgeon-General of the United States, grotesquely exaggerate the consequences of smoking in deaths from cancer and CHD, and the virtues of giving up smoking, it is small wonder that the media, as well as politicians, take up the chorus, not realizing that there is little justification for the claims made, and that they have received damaging criticism from leading oncologists, immunologists, and other scientists.

The only responsible way out of this practice, which is seriously embarrassing for medicine and science alike, is to return to the precepts which have always guided science in the past. Publications should contain not only assertions of position, based on selected and biased evidence, but should deal fairly and squarely with all objections, criticisms and contrary findings. Conclusions should be justified by the evidence, and carefully qualified rather than extreme in their certainty when this is not warranted. Finally, no publicity should be given to results, and no publication urged or taken, unless there is virtual agreement among experts (as there most certainly is not in relation to the effects of smoking) about the issue in question. Even then the fallability of research in such very complex areas should be emphasized, and the possibility of error acknowledged. These are the ways science has progressed, and has acquired its unique prestige; we should be careful not to squander it in the way that it has been squandered in relation to these controversies.

What is particularly to be regretted is the intrusion of political associations into scientific debates. It is interesting to note that one of the most determined protagonists of the "smoking causes disease" view, (Everett Koop, the former U.S. Surgeon-General)

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has recently been condemned by a House of Representative Committee for suppressing evidence on another issue, also involving social and ethical considerations, namely abortion. According to the Committee, Koop refused to publish a study of the physical and psychological effects of abortion on women, because the study found no evidence that the procedure harms women. At least one scientist working at the U.S. Government's Center for Disease Control, whose research focused on abortion, had his findings censored, and another was demoted, according to the Committee. Apparently when the Report commissioned by the White House had failed to provide the hoped-for evidence condemning abortion, "he therefore decided not to issue a report, but instead to write a letter to the president which would be sufficiently vague as to avoid supporting the pro-choice position that abortion is safe for women." Federal funding of research on contraception had halved since 1980, Government officials often treated those who did research on abortion with hostility, and officials who did not obey the anti-abortion line and dared to contradict Koop were fired or transferred, and their writing censored.

These are precisely the tactics that have been used widely in connection with the "smoking causes disease" issue. It has become difficult for those who wish to examine the problem objectively to obtain research funds, or to publish their data, if they are not in line with official policy. Newspapers refuse to discuss the facts objectively, and pretend that unanimity exists when in reality confusion reigns, and criticism of the "orthodox" view is widespread. Alternative models are dismissed without proper examination, and are never mentioned in official publications. Investigators who show an interest in such models encounter obstacles in their careers, and may have all support withdrawn, regardless of the quality of their

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research. These are not conditions which encourage high-class scientific research, and it is small wonder that the field is confused and full of anomalies. In this book I have tried to set out the facts as carefully and objectively as possible, and to point out the major conclusions which may be drawn.

The failure of many workers in this field to be objective, and to tell the public fairly and squarely about the present position of research, has been exposed most convincingly by Effron (1984) in her book on "The Apocalyptics", which cuts a much wider swathe than the present book, but which justifies amply its subtitle: "Cancer and the big lie". It deals with "the ideological corruption of cancer research in the United States." The author contends, and demonstrates, that an extreme environmental movement, which she calls "the apocalyptics", has politically distorted research in environmental cancer, saturating the United States with theories of cancer that are pure myths. Her book should be read by all concerned with the problem of cancer origin and propagation; the problem considered in this book is only one facet of this much wider phenomenon. There is no doubt that smoking is one of many risk factors in cancer and CHD, but the notion that it is all-important, and that reduction in smoking would have far-reaching benevolent consequences as far as cancer and CHD are concerned, is just one of those myths which Effron castigates.

There is much that remains obscure, but there are also some features of the scene which must be regarded as hopeful. If stress and other psychosocial factors are indeed killers, it does seem that suitable prophylactic means are at hand to delay or prevent cancer and coronary heart disease. Behaviour therapy is a very cheap and convenient way of safeguarding healthy probands from cancer and

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coronary heart disease, and from every point of view prevention must be better than largely non-existent cures or very expensive treatments with often disastrous side-effects. If only we could disabuse ourselves of the present unhealthy preoccupation with smoking, and turn our attention to the real enemy, namely stress, we could in reality save all those lives which a dishonest propaganda effort has tried to persuade us could be saved if only we gave up smoking. This is a serious issue, vital for hundreds of thousands of people, and it is irresponsible to continue a bland and biased disregard of the true facts of the case.

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Factor Intervention Trial Study was stated to have cost 115 million dollars, with the true cost being probably appreciably higher; yet that study was fundamentally flawed from the beginning, was badly executed and analysed, and gave results that were embarrassing to the medical profession as a whole. Had the money been spent on proper scientific research we would be very much more knowledgeable than we are now.

In particular, research is vital in the area we know least about, namely the effects of stress and of behaviour therapy on the immune system; prospective studies of the kind pioneered by Grossarth-Maticek (see references) should be repeated with the inclusion of direct measures of immune system assays, and with multiple measures of sclerosis. Only in this way will we achieve greater knowledge of the causes of cancer and coronary heart disease, and of the most efficient methods of preventing them.

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CAPTIONS

Table 1. Comparison of CHD morbidity ratios and incidence rates of never-smokers and ex-smokers at various years of follow-up. (After Seltzer, 1989.)

" 2. CHD risk ratios (cigarette smokers/non-smokers) calculated from the Framingham Heart Study incidence data. (After Seltzer, 1989.)

" 3. Comparison of U.S. Surgeon-General's coronary heart disease findings and those of the Framingham Study data with respect to cigarette smoking. (After Seltzer, 1989.)

" 4. Causes of death for different types of probands - Yugoslav sample. (Grossarth-Maticek & Eysenck, 1980.)

" 5. Causes of death for different types of probands - Heidelberg normal sample. (Grossarth-Maticek & Eysenck, 1988.)

" 6. Causes of death for different types of probands - Heidelberg stressed sample. (Grossarth-Maticek & Eysenck, 1988.)

" 7. Interaction between smoking, personality and death from lung cancer. (Grossarth-Maticek & Eysenck, 1988.)

" 8. Relative importance of personality and organic risk factors for death. (Grossarth-Maticek, Eysenck & Vetter, 1988.)

" 9. Synergistic effects of H (heredity), B (chronic bronchitis), C (cigarette smoking) and S (stress/personality), BT (behaviour therapy.) (Grossarth-Maticek & Eysenck, in press.)

" 10. Effects of behaviour therapy on cancer and coronary heart disease. (Grossarth-Maticek & Eysenck, 1989.)

" 11. Effects of group behaviour therapy on cancer and coronary heart disease. (Grossarth-Maticek & Eysenck, 1989.)

" 12. Effects of bibliotherapy and short behaviour therapy on cancer and coronary heart disease. (Grossarth-Maticek & Eysenck, 1989.)

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TABLE 1.

Follow-up.	Morbidity ratios* or incidence rate		Proportionate difference
9.1 yr	Never smokers	77	+67%
	Past "	46	
12-yr ("heart attacks")	Never smokers	82	+39%
	Ex-smokers	59	
16-yr	Never smokers	91	+49%
	Ex-smokers	61	
18-yr ("heart attacks")	Never smokers	17.7~	+36%
	Ex-smokers	13.0~	
22-yr	Never smokers	29.8~	+23%
	Ex-smokers	24.2~	

*Ratio of observed to expected rate (100 times those observed divided by those expected).
~CHD incidence rates per 100 men.

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TABLE 2

Follow-up	Incidence	Ages	Nonsmoker classification for for comparison with smokers^	Risk Ratios Men	Women
9.1 yr	After Exam 1	40-59	Never smokers	1.5~	-
			Nonsmokers (never, past, <half day)	1.8~	-
12-yr	After Exam 1	30-59	Non-cigarette	1.8|	1.0|
14-yr	After Exam 1	35-64	None	1.4	0.9
16-yr	After Exam 1	35-64	None	1.3	0.9
	Average annual incidence rate	35-74	None	1.3	0.8
18-yr	Average annual incidence rate	45-74	None	1.3	1.0
20-yr	Average annual "smoothed" rate	45-74	None	1.4	-
22-yr	After Exam 1	30-59	Non-cigarette	1.3	0.8
24-yr	After Exam 1	30-59	Non-cigarette	1.3	0.9
26-yr	After Exam 1	35-84	Non-cigarette	1.3@	-
30-yr	Average annual incidence rate	35-64	None	1.6	1.1
		35-74		1.3	1.0
		35-84		1.0	1.0

* Age-adjusted by assigning equal weights to rates in each age-group, in accordaince with Framingham method listed in Table 1.

^ Framingham categories of "none" and "non-cigarette" include ex-smokers, and pipe and cigar smokers in addition to "never smokers".

~ "Standardized incidence ratios" with CHD limited to non-fatal and fatal MI, and sudden CHD death.

| Framingham "morbidity ratios".

@ Framingham "coronary morbidity".

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TABLE 3

Criteria	U.S. Surgeon-General's findings	Framingham data findings
Men-Univariate association	Strong	Weak
Men-Multivariate "	Strong	Non-significant; absent
Women-Univariate "	Present, but less than in men	Absent
Women-Multivariate "	Present	Absent
Duration of cigarette smoking	Increase of CHD with increase of duration of smoking	Absent
Cessation of cigarette smoking	Gradual reduction of CHD to level of non-smokers	Immediate reduction of CHD to level below that of never smokers.

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Table 4.

Yugoslavia	Living	Cancer	Coronary heart disease	Other causes of deaths	Total N
Type 1	72 = 23.8%	140 = 46.2%	23 = 8.3%	66 = 21.8%	303
Type 2	96 = 28.3%	19 = 5.6%	99 = 29.2%	125 = 36.9%	339
Type 3	123 = 56.7%	4 = 1.8%	20 = 9.2%	70 = 32.3%	217
Type 4	437 = 90.7%	3 = 0.6%	8 = 1.7%	34 = 7.1%	482
Impossible to allocate to type	6	0	4	2	12
Total	734 = 54.2%	166 = 12.3%	156 = 11.5%	297 = 27.0%	1353

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Table 5.

Heidelberg normal	Living	Cancer	Coronary heart disease	Other causes of deaths	Total N
Type 1	78 = 71.6%	19 = 17.4%	2 = 1.8%	10 = 9.2%	109
Type 2	109 = 64.1%	10 = 5.9%	23 = 13.5%	28 = 16.5%	170
Type 3	185 = 98.4%	0	1 = 0.5%	2 = 1.1%	188
Type 4	387 = 99.0%	0	1 = 0.3%	3 = 0.8%	391
Impossible to allocate to type	14	0	0	0	14
Total	773 = 88.6%	29 = 3.3%	27 = 3.1%	43 = 4.9%	872

2021611041

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

Table 6.

Heidelberg 'stressed' group	Living	Cancer	Coronary heart disease	Other causes of death	Total *N*
Type 1	188 = 38.4%	188 = 38.4%	34 = 7.0%	79 = 16.2%	489
Type 2	148 = 47.9%	7 = 2.3%	86 = 27.8%	68 = 22.0%	309
Type 3	153 = 92.7%	4 = 2.4%	0	8 = 4.8%	165
Type 4	71 = 97.3%	0	0	2 = 2.7%	73
Impossible to allocate to type	6	0	0	0	6
Total	566 = 54.3%	199 = 19.1%	120 = 11.5%	157 = 15.1%	1042

2021611042

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

Table 7.

		Yugoslavia			Heidelberg (stressed)		
		Lung cancer deaths	Other deaths	Total	Lung cancer deaths	Other deaths	Total
Non-smokers	Type I	1 = 0.8%	118	119	9 = 3.8%	227	236
	Others	0	550	550	3 = 1.0%	297	300
Smokers	Type I	31 = 16.9%	153	184	37 = 14.6%	216	253
	Others	6 = 1.2%	482	488	0	247	247

2021611043

TABLE 8.

	Type	Mean Y	Mean HI	Mean H2	b Y	b HI	b H2	Mort. (per cent) Y	Mort. (per cent) HI	Mort. (per cent) H2
rf: systolic blood pressure	1	151.8	—	174.2	0.056	—	0.024	7.6	—	7.2
dis: infarct/stroke mortality	2	160.7	—	207.6	0.084	—	0.108	27.2	—	23.7
	3	148.3	—	186.3	0.005	—	0.010	7.7	—	2.3
	4	144.6	—	185.8	0.003	—	0.021	1.8	—	5.0
	all	150.7	—	187.7	0.035	—	0.041	11.1	—	9.1
Significance of differences		0.0000	—	0.0000	0.0004	—	0.0011	<0.0001	—	<0.0001
rf : diastolic blood pressure	1	90.8	—	85.8	0.026	—	0.019	5.1	—	7.2
dis.: infarct/stroke mortality	2	93.7	—	93.5	0.093	—	0.071	26.8	—	24.5
	3	88.6	—	88.9	0.030	—	0.004	8.5	—	2.5
	4	86.8	—	89.0	0.014	—	0.007	2.1	—	5.5
	all	89.6	—	88.8	0.038	—	0.025	10.6	—	9.9
Significance of differences		0.0000	—	0.0000	0.0137	—	0.0494	<0.0001	—	<0.0001
rf: blood cholesterol	1	255.6	217.5	258.3	0.011	0.004	0.037	8.3	3.2	8.5
dis.: infarct/stroke mortality	2	250.4	254.4	305.1	0.036	0.054	0.046	29.4	9.7	24.7
	3	245.8	216.8	282.6	0.027	0.000	0.003	9.2	1.8	1.8
	4	245.5	217.9	280.5	0.001	0.001	0.000	1.8	0.8	3.5
	all	249.1	224.9	277.8	0.019	0.014	0.020	12.2	3.9	9.6
Significance of differences		0.0330	0.0000	0.0000	NS	NS	0.0486	<0.0001	0.0008	0.0000
rf: cigarettes per day	1	15.7	13.0	16.9	0.000	0.003	0.000	7.7	4.1	6.9
dis.: infarct/stroke mortality	2	10.4	18.8	14.4	0.038	0.050	0.161	29.8	17.0	27.6
	3	11.6	8.9	12.2	0.021	0.016	0.007	8.3	2.5	1.7
	4	10.8	8.2	10.9	0.002	0.008	0.011	1.8	1.2	3.5
	all	11.9	11.1	15.0	0.005	0.019	0.045	11.9	5.8	9.9
Significance of differences		0.0000	0.0000	0.0000	NS	NS	0.0000	<0.0001	0.0002	0.0001
rf: cigarettes per day	1	15.7	13.0	16.9	0.075	0.019	0.044	8.3	3.6	8.3
dis.: lung cancer mortality	2	10.4	18.8	14.4	0.020	0.010	0.003	2.5	2.5	1.5
	3	11.6	8.9	12.2	0.003	0.015	0.007	1.3	2.4	1.7
	4	10.8	8.2	10.9	0.002	0.008	0.011	0.6	1.2	3.5
	all	11.9	11.1	15.0	0.025	0.013	0.016	3.1	2.4	3.7
Significance of differences		0.0000	0.0000	0.0000	0.0001	NS	0.0113	<0.0001	NS	0.0000

largest (abs.) value is underlined

For Yugoslavia, the organic variables represent a single measurement taken in 1966 (cholesterol: 1969).
For Heidelberg, the organic variables are the average of up to 7 measurements taken in 1972.
Abbreviations:

rf = risk factor.
dis. = disease.
mean = mean of organic variable within type groups.
b = regression coefficient of the dependent variable on the organic variable within type groups.
mort. = mortality (the dependent variable) within type groups, adjusted for the organic variable.
Y = Yugoslavia HI = Heidelberg representative H2 = Heidelberg stressed. NS means $P > 0.05$.

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

2021611044

Table 9.

Combination of risks:	N	Lung cancer	%	Other causes of death	%	Average Age
Only H	50	0	0	5	10	51
Only C	100	0	0	12	12	52
Only S	59	0	0	16	27	52
H + C	50	1	2	4	8	53
H + B	52	0	0	8	15	51
C + B	55	0	0	11	20	52
C + S	100	2	2	21	21	53
H + S	49	0	0	9	18	54
B + S	50	0	0	8	16	53
C + H + B	26	2	8	5	19	51
C + H + S	50	10	20	14	28	51
C + B + S	51	5	10	10	20	51
H + C + B + S	26	8	31	8	31	52
(H+C+B+S+BT)	26	3	12	4	15	52

2021611045

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

Table 10.

I.	N	CANCER Deaths	CANCER Incidence	Other causes of death	Living
Control	50	16 32%	21 42%	15 30%	19 38%
Therapy	50	0 0	13 26%	5 10%	45 90%
TOTAL:	100	16 16%	34 34%	20 20%	64 64%

II.	N	CHD Deaths	CHD Incidence	Other causes of death	Living
Control	46	16 34.8%	20 43.5%	13 28.3%	17 36.9%
Therapy	46	3 6.5%	11 23.9%	6 13%	37 80.4%
TOTAL:	92	19 20.6%	31 33.7%	19 20.7%	54 58.7%

2021611046

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

Table 11.

N Not contacted	Therapy 245 6		Control 245 11	
	Mortality	Incidence	Mortality	Incidence
	239	235	234	231
Cancer	18 7.5%	75 31.9%	111 47.4%	129 55.8%
CHD	10 4.2%	29 12.3%	36 15.4%	45 19.5%
Other causes of death	20 8.4%	-	33 14.1%	-
Living	191 79.9%		56 23.9%	

2021611047

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

Table 12.

	Causes of Death									
	Cancer		CHD		Other					
	D*	I*	D*	I*	D*	I*	Total	Living	Not investigated	
Control N=500	106 21.5%	162 33.4%	145 29.4%	203 41.8%	164 33.3%	-	415 84.2%	78 15.8%	7 1.4%	15 3%
Control with use of psychoanalytic text. (Placebo Group.) N=100	22 22%	37 37.7%	31 31%	40 40.8%	28 28%	-	81 81%	19 19%	0 0%	2 2%
Therapy Group with Behaviour Therapy Text. N=600	27 4.5%	99 16.9%	47 7.9%	132 22.5%	115 19.2%	-	189 31.6%	409 68.4%	2 0.3%	14 2.3%

* D = died; I = incidence

2021611048

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

Fig. 1. Cumulative experience of intercourse by age of first intercourse for college educated males and females in the USA. (Data from the National Longitudinal Study.)

" 2. Effect of different types of treatment on giving up smoking. (O'Connor & Stravynski, 1982.)

" 3. Personality type and cause of death, Yugoslav study. (Grossarth-Maticek & Eysenck, 1988.)

" 4. Personality type and cause of death, Heidelberg normal sample. (Grossarth-Maticek & Eysenck, 1988.)

" 5. Personality type and cause of death, Heidelberg stressed sample. (Grossarth-Maticek & Eysenck, 1988.)

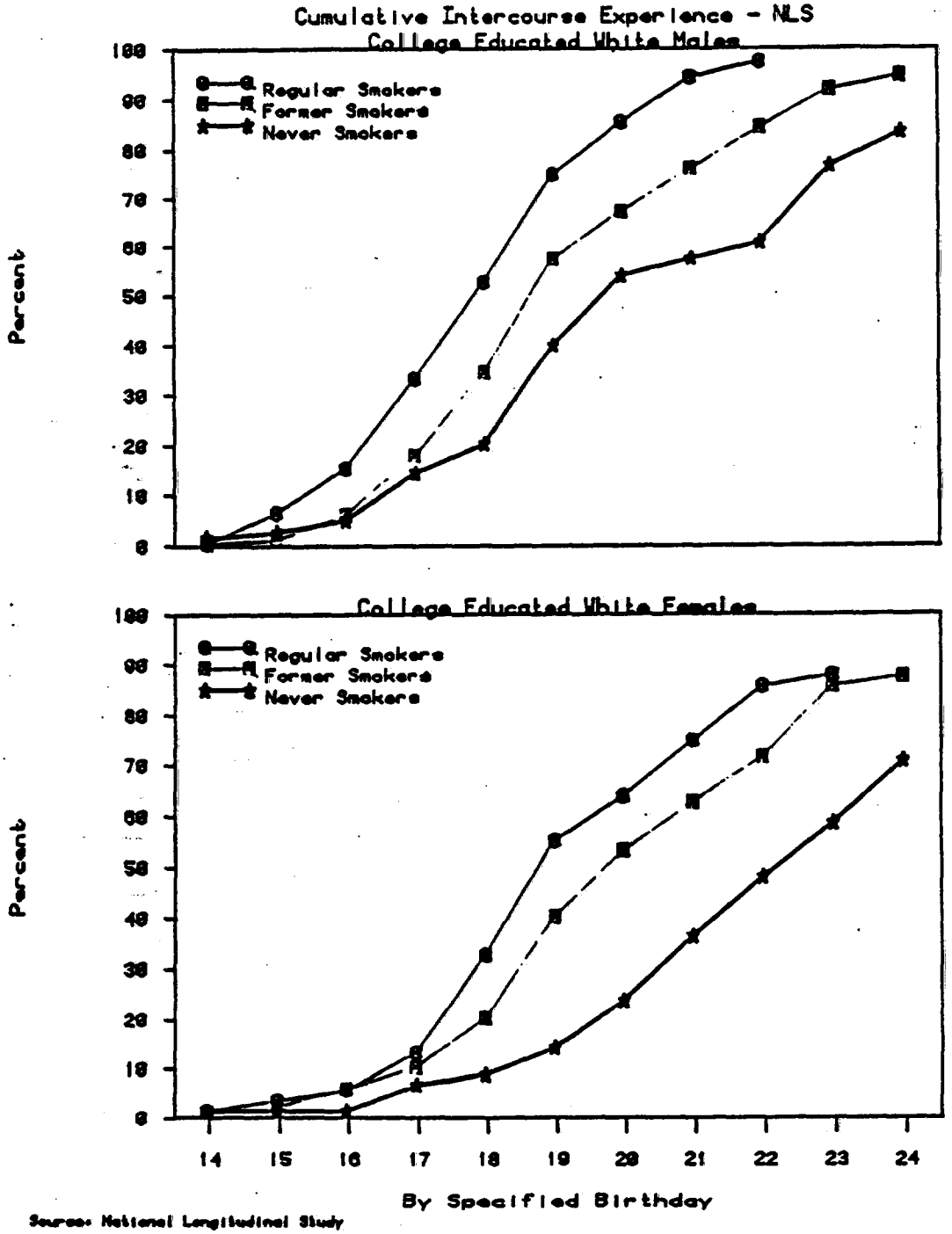
" 6. Synergistic action of 7 risk factors for cancer. (Grossarth-Maticek & Eysenck, in press a.)

" 7. Sclerosis in cancer-prone and coronary heart disease-prone probands and effects of behaviour therapy. (Grossarth-Maticek & Eysenck, in press b.)

2021611049

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

FIG. 1

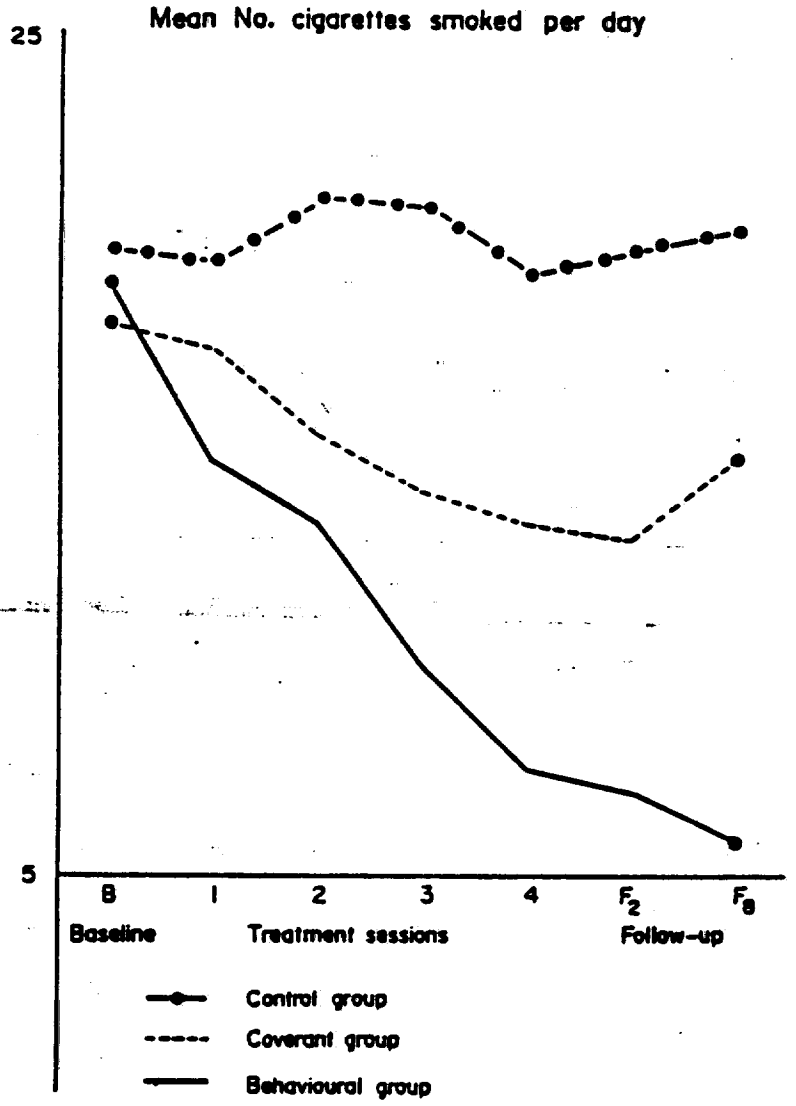


Source: National Longitudinal Study

2021611050

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

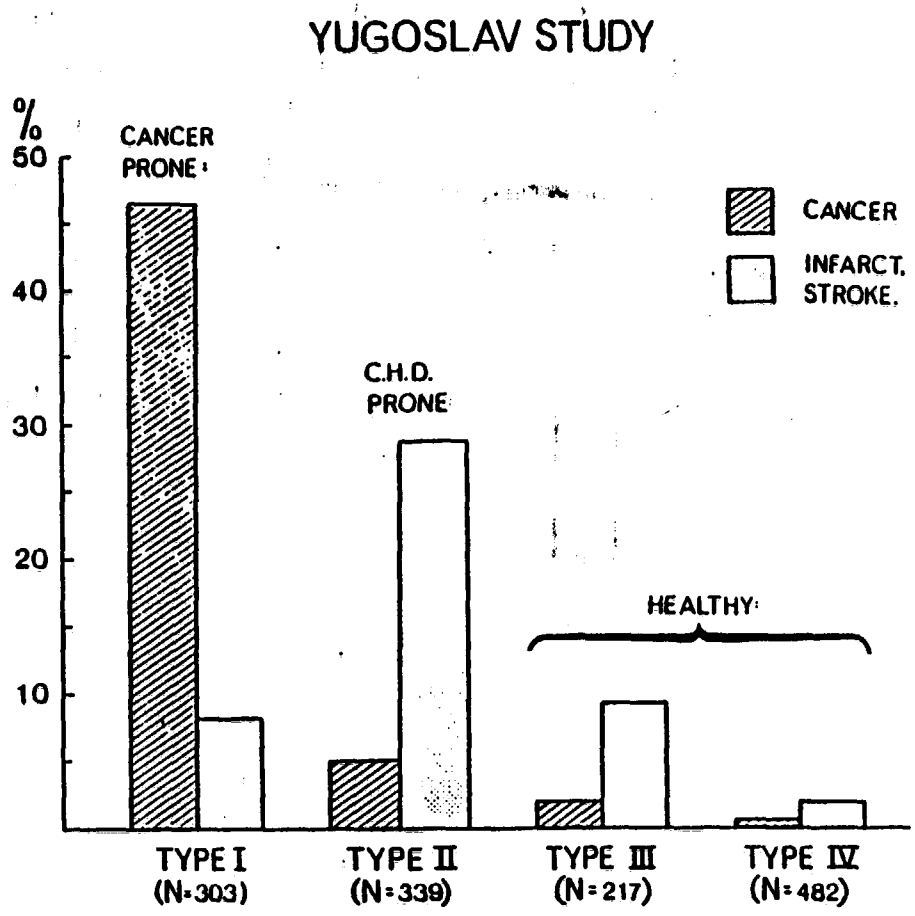
FIG. 2.



2021611051

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

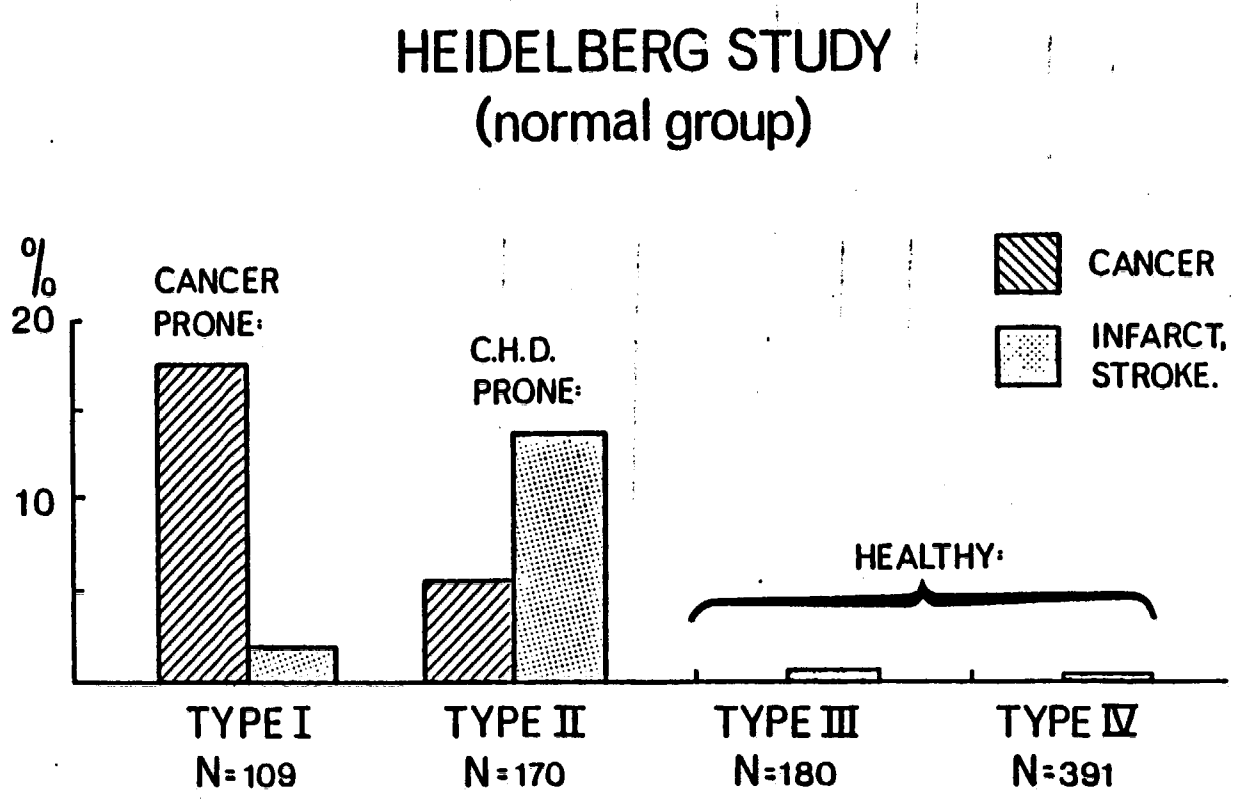
FIG. 3



20216110S2

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

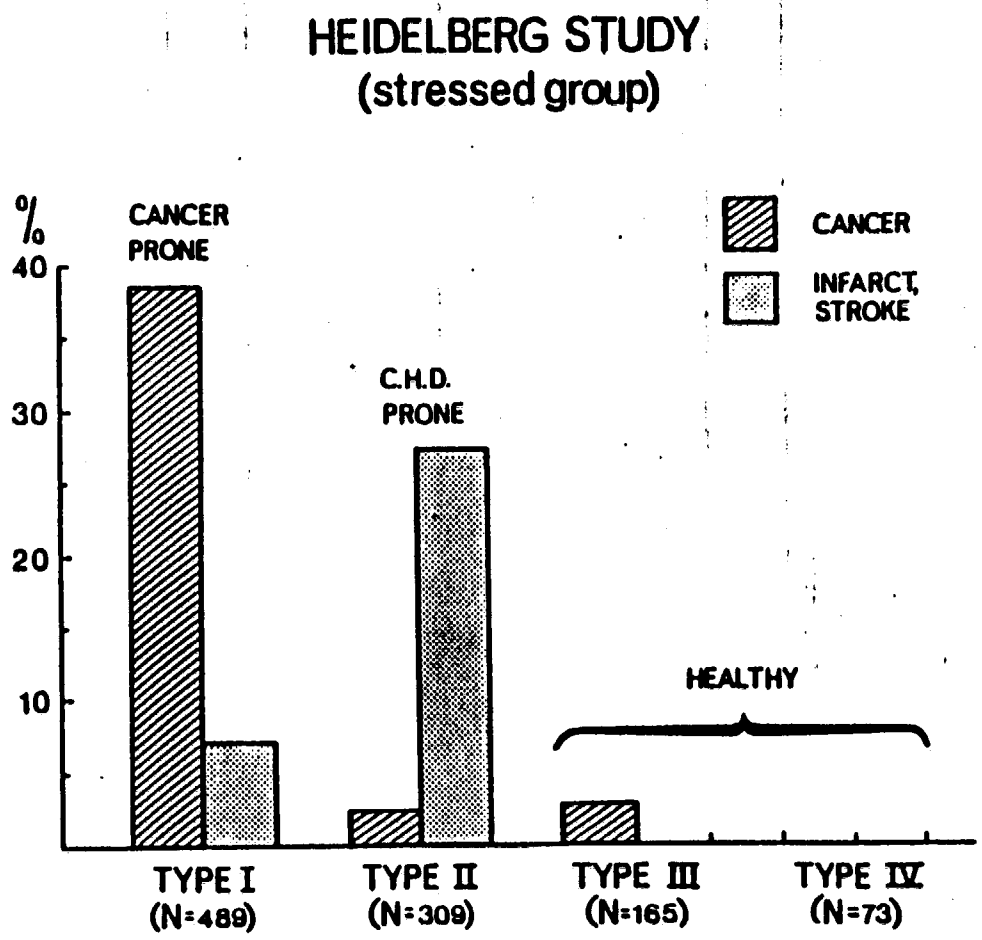
FIG. 4.



2021611053

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

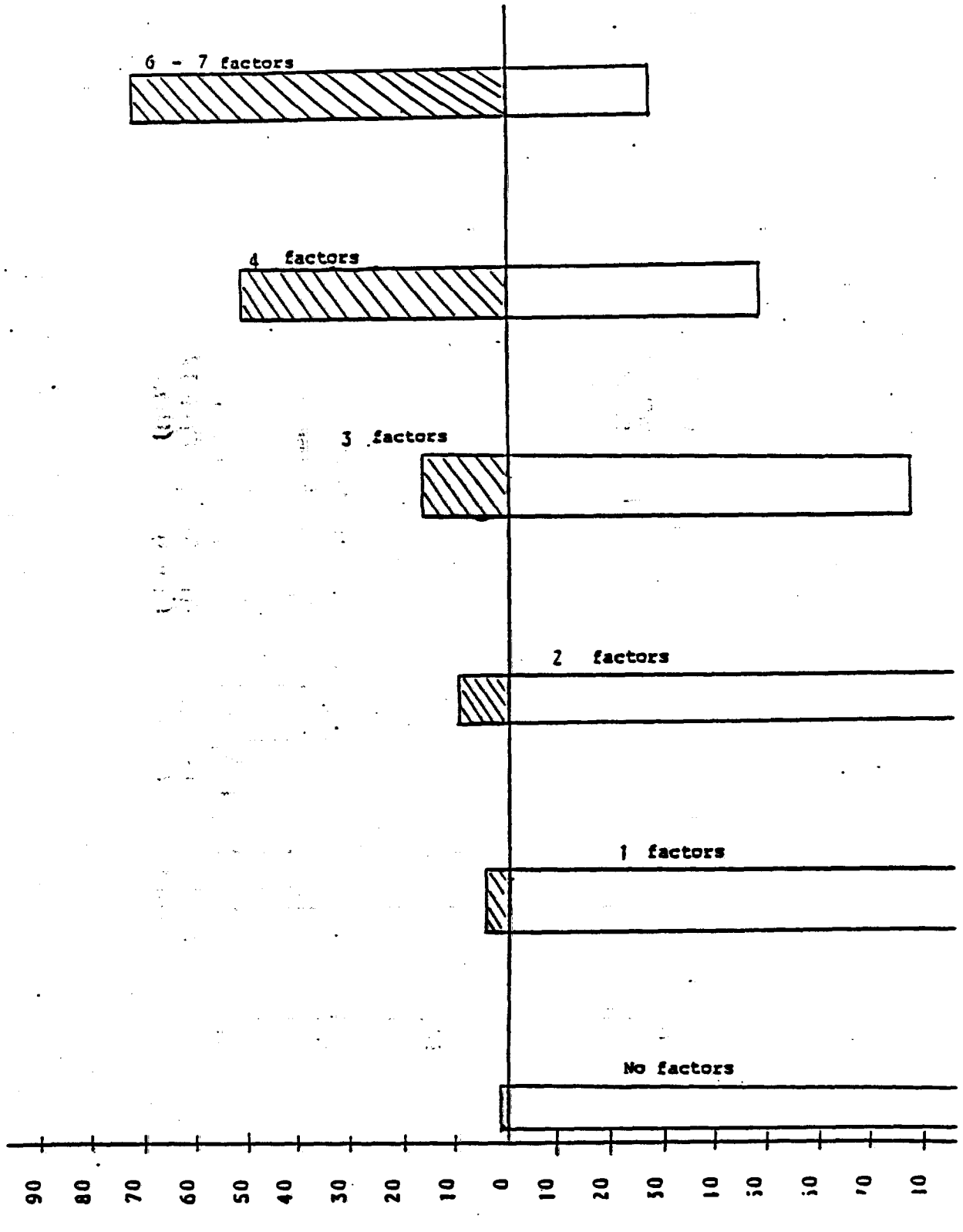
FIG. 5.



2021611054

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

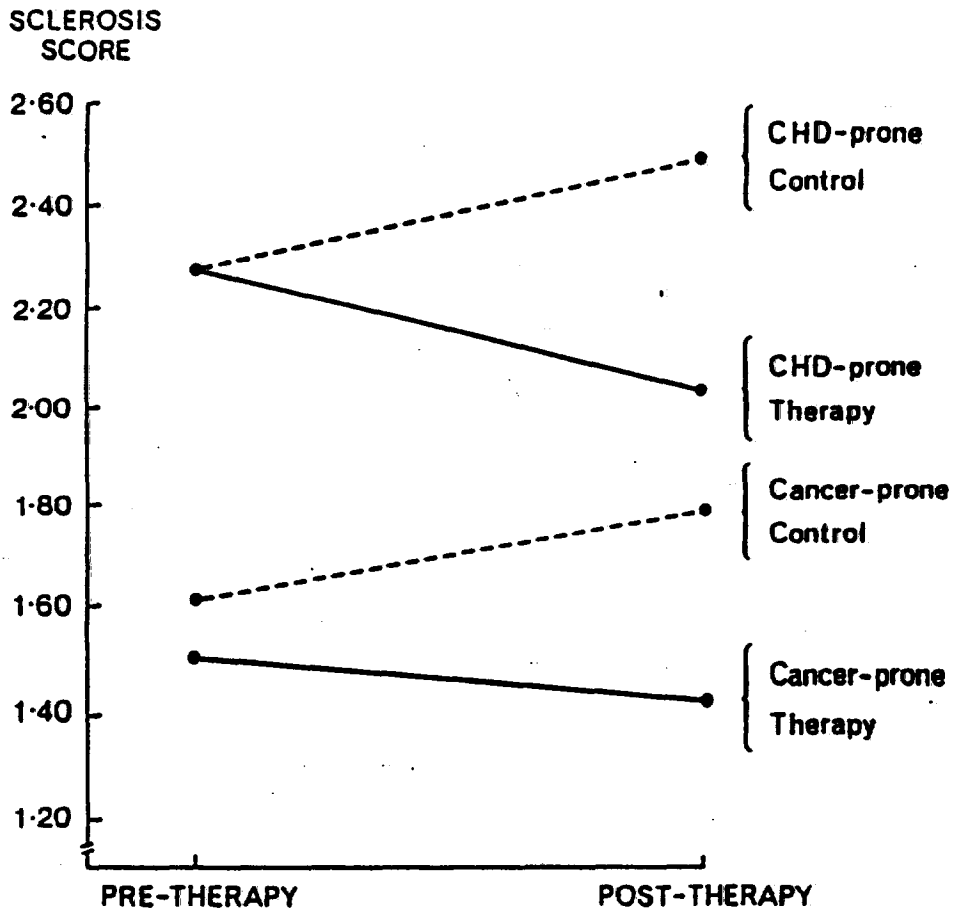
Fig. 6.



2021611055

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

FIG. 7.



2021611056

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

APPENDIX

SHORT DISEASE-PRONENESS INVENTORY

...

(For scoring, see instructions at end of inventory)

1) I find it very difficult to stand up for myself. YES NO

2) I have been complaining for years about various unfavourable conditions, but am not able to change them. YES NO

3) I am mainly concerned with my own well-being. YES NO

4) I am usually content and happy with my daily activities. YES NO

5) I can express my feelings only when there are good reasons for them. YES NO

6) I don't believe in social rules, and don't pay much attention to other people's expectations or the obligations I may have towards them. YES NO

7) I cannot live happily and contentedly with nor without a particular person. YES NO

8) I prefer to agree with others, rather than assert my own views. YES NO

9) Certain people are the most important causes

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Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

of my personal misfortunes. YES NO

10) I alternate to a great degree between the positive and negative evaluation of people and conditions. YES NO

11) When I cannot achieve closeness with someone who is emotionally important to me, I have no difficulties in letting them go. YES NO

12) I have difficulties in showing my emotions because for every positive emotion there is a negative one. YES NO

13) My behaviour towards other people alters from being very friendly and good-natured to being very hostile and aggressive. YES NO

14) I cannot live happily and contentedly in the presence or the absence of certain states or conditions; e.g. I need my work, but am unhappy doing it. YES NO

15) I tend to act more to fulfill the expectations of people close to me, rather than look after my own needs. YES NO

16) Certain conditions or situations are the most important causes of my personal misfortunes. YES NO

17) With people I love, I keep changing from keeping them at a great distance to stifling dependence, and from stifling dependence to

2021611058

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

excessive distancing. YES NO

18) I can usually arrange things so that people who are emotionally important to me are as close to or as distant from me as I wish. YES NO

19) Reason, rather than emotion, guides my behaviour. YES NO

20) I often expect others to fulfill agreements very strictly, but do not believe in doing so myself. YES NO

21) I often have thoughts which terrify me and make me unhappy. YES NO

22) I tend to give in and abandon my own aims in order to achieve harmony with other people. YES NO

23) I feel helpless against people or conditions which cause great unhappiness for me, because I cannot change them. YES NO

24) When I am in a situation which I experience as threatening, I immediately try to get other people to help and support me. YES NO

25) When I fail to achieve my objectives, I can easily change tack. YES NO

26) When people make emotional demands on me, I usually react only rationally, never emotionally. YES NO

27) I usually act in a spontaneous manner, following my immediate feelings without

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Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

considering the actual consequences. YES NO

28) Relations with certain people are always pretty unsatisfactory, but there is nothing I can do about it. YES NO

29) I am unable to express my feelings and needs openly to other people. YES NO

30) I always seem to be confronted with the undesirable aspects of people and conditions. YES NO

31) When someone who is emotionally important to me hurts me ever so slightly, I immediately dissociate myself from that person. YES NO

32) I can manage to live fairly contentedly with or without someone who is emotionally important to me. YES NO

33) I am quite unable to allow myself to be guided by emotional considerations. YES NO

34) I often feel like attacking other people and crushing them. YES NO

35) Certain situations and states (e.g. at my place of work) tend to make me unhappy, but there is nothing I can do to alter things. YES NO

36) I tend to accept conditions which work against my personal interests without being able to protest. YES NO

37) Certain people keep interfering with my

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Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

personal development. YES NO

38) I expect others to live up to the highest moral standards, but do not feel that these are binding on myself. YES NO

39) I can usually change my behaviour to suit conditions. YES NO

40) My actions are never influenced by emotions to the degree that they might appear irrational. YES NO

41) When my partner demonstrates love towards me, I sometimes become particularly aggressive. YES NO

42) Certain bodily conditions (e.g. being overweight) make me unhappy, but I feel unable to do anything about it. YES NO

43) I often feel inhibited when it comes to openly showing negative feelings such as hatred, aggression, or anger. YES NO

44) Certain conditions keep interfering with my personal development. YES NO

45) I seek satisfaction of my own needs and desires first, regardless of the needs and rights of others. YES NO

46) I am usually capable of finding new points of view and successful, sometimes surprising,

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Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

solutions for problems. YES NO

47) I always try to do what is rational and logically correct. YES NO

48) When I feel like attacking someone physically, I have no inhibitions about doing this at all. YES NO

49) I can relax bodily and mentally only very rarely; most of the time I am very tense. YES NO

50) I am inclined not to be demonstrative when emotional shocks upset me. YES NO

51) I cannot control excitement or stress in my life because this is dependent on the actions of other people. YES NO

52) When I make emotional demands on another person, I require immediate satisfaction. YES NO

53) I am independent in what I do, and do not depend on other people when this works to my disadvantage. YES NO

54) I always try to express my needs and desires in a rational and reasonable manner. YES NO

55) I have no inhibitions in hurting myself physically if I feel like doing so. YES NO

56) I have great difficulties in entering into happy and contended relations with people. YES NO

57) When I feel emotionally let down I tend to be

2021611062

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

paralysed and inhibited. YES NO

58) I cannot control excitement or stress in my life because this depends on conditions over which I have no control. YES NO

59) I usually find fulfilment in everyday situations which are not subject to ordinary rules, regulations and expectations. YES NO

60) When things don't work out, this does not make me give up, but rather makes me change my way of doing things. YES NO

61) I try to solve my problems in the light of relevant and rational consideration. YES NO

62) I resent all moral obligations because they hamper and inhibit me. YES NO

63) I am helpless when confronted with emotional shocks, depression or anxiety. YES NO

64) When something terrible happens to me, such as the death of a loved one, I am quite unable to express my emotions and desires. YES NO

65) I can express my aims and desires clearly but feel that it is quite impossible to achieve them. YES NO

66) As soon as someone becomes emotionally

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Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

important for me, I tend to place contradictory demands upon them, such as "Don't ever leave me" or "Get away from me." YES NO

67) When things lead to harmful results for me, I have no trouble in changing my behaviour to make for success. YES NO

68) I only believe in things which can be proved scientifically and logically. YES NO

69) When it benefits me I have no hesitation in lying and pretending. YES NO

70) I am seldom able to feel enthusiasm for anything. YES NO

2021611064

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000

Scoring Instructions for Short Disease-Proneness Inventory:

Type 1: Add "YES" answers to questions:

1, 8, 15, 22, 29, 36, 43, 50, 57, 64.

Type 2 Add "YES" answers to questions:

2, 9, 16, 23, 30, 37, 44, 51, 58, 65.

Type 3: Add "YES" answers to questions:

3, 10, 17, 24, 31, 38, 45, 52, 59, 66.

Type 4a: Add "YES" answers to questions:

4, 11, 18, 25, 32, 39, 46, 53, 60, 67.

Type 4b: Add "NO" answers to questions:

7, 14, 21, 28, 35, 42, 49, 56, 63, 70.

Type 5: Add "YES" answers to questions:

5, 12, 19, 26, 33, 40, 47, 54, 61, 68.

Type 6: Add "YES" answers to questions:

6, 13, 20, 27, 34, 41, 48, 55, 62, 69.

To decide on TYPE, add 4a and 4b, and divide by 2.
A person's type is then the one on which he has the highest score. This is the ipsative way of scoring.

Another method is to take all 6 scores into account:

Type 1 is the cancer-prone type;
" 2 " " coronary heart disease-type;
" 3 " " alternating reaction type - reasonably healthy;
" 4 " " autonomous, healthy type;
" 5 " " rational-antiemotional type; and
" 6 " " anti-social, egocentric type.

(For further details, see Grossarth-Maticek & Eysenck (1990).

2021611065

Source: https://www.industrydocuments.ucsf.edu/docs/pjvl0000